## Title

Diet Quality Scores Inversely Associated with Postmenopausal Breast Cancer Risk Are Not Associated with Premenopausal Breast Cancer Risk in the California Teachers Study.



**Journal**
The Journal of nutrition, 148(11)

**ISSN**
0022-3166

**Authors**
Haridass, Vikram
Ziogas, Argyrios
Neuhausen, Susan L
et al.

**Publication Date**
2018-11-01

**DOI**
10.1093/jn/nxy187

# Diet Quality Scores Inversely Associated with Postmenopausal Breast Cancer Risk Are Not Associated with Premenopausal Breast Cancer Risk in the California Teachers Study

Vikram Haridass,[1] Argyrios Ziogas,[1] Susan L Neuhausen,[2] Hoda Anton-Culver,[1] and Andrew O Odegaard[1]

[1]Department of Epidemiology, School of Medicine, University of California, Irvine, CA and [2]Department of Population Sciences, Beckman Research Institute of City of Hope, Duarte, CA

**Abstract**

**Background:** Evidence for the association between diet and breast cancer risk is inconsistent Thus, research that compares indexes of overall diet quality may provide new insight.

**Objective:** We examined the association between diet quality indexes and pre- and postmenopausal breast cancer risk in a large prospective cohort.

**Methods:** This was a prospective analysis of 96,959 women, aged 22–104 y, in the California Teachers Study cohort (1995–2011). Diet quality was characterized by 4 different indexes. Specifically, we examined Alternate Mediterranean Diet (aMED), Alternative Healthy Eating Index–2010 (AHEI-2010), Dietary Approaches to Stop Hypertension (DASH), and Paleolithic index (PALEO) scores with the risk of developing breast cancer. We used multivariable Cox proportional hazards regression models to derive HRs and 95% CIs for breast cancer risk.

**Results:** In the analysis of 42,517 women at risk of premenopausal breast cancer, there was no association between any of the indexes and incident breast cancer (346 cases). In the analysis of 54,442 women at risk of postmenopausal breast cancer at baseline, higher AHEI-2010, aMED, and DASH scores were inversely associated with incident breast cancer (3523 incident cases). Respectively, HRs (95% CIs) comparing quintile 5 to quintile 1 (reference) for AHEI-2010, aMED, and DASH indexes were 0.87 (0.78, 0.97; *P*-trend = 0.004), 0.91 (0.82, 1.02; *P*-trend = 0.03), and 0.89 (0.80, 1.00; *P*-trend = 0.03). The PALEO score was not associated with postmenopausal breast cancer (HR for quintile 5 compared with quintile 1: 1.05; 95% CI: 0.94, 1.17).

**Conclusions:** Diet quality indexes that emphasize intake of whole grains, vegetables, fruits, legumes, and nuts and seeds and de-emphasize red and processed meats and sugar-sweetened beverages were modestly associated with a lower risk of incident postmenopausal breast cancer risk. However, they were not associated with premenopausal breast cancer, and the PALEO score was not associated with cancer risk regardless of menopausal status.

## Introduction

In the United States, breast cancer is the most common cancer in women, with ∼1 in 8 expected to develop the cancer in their lifetime (1). Research addressing modifiable breast cancer risk factors has the potential to lower this rate by improving recommendations, policy, and advocacy. Dietary intake is considered a risk factor for breast cancer, but the evidence informing this topic is inconsistent. Some studies reported an association between dietary patterns and breast cancer risk (2–7), and others observed no association (8, 9), with results also differing by menopausal status (10, 11). Indeed, these results may be partially explained by different methodologic approaches (8). Yet, systematic reviews suggest that a dietary pattern with higher intakes of fruits, vegetables, whole grains, nuts, and legumes and lower intakes of sugar-sweetened beverages and red and processed meats was inversely associated with breast cancer risk (9), although some studies did not observe an inverse association (7, 11). This evidence base aligns with the 2015 US Dietary Guidelines for Americans, which recommend focusing on the overall diet rather than on individual nutrients (12), and these guidelines generally align with dietary recommendations for cancer prevention (9, 13, 14). In addition, the 2015 Dietary Guidelines for Americans scientific report noted the need to refine these dietary recommendations for breast cancer risk and called for further research examining overall dietary patterns and the need to consider both pre- and postmenopausal breast cancer risks separately due to the hormone-dependent nature of breast cancer (12, 14).

Therefore, to contribute to the noted evidence gap, we calculated scores for 3 established a priori diet quality indexes with a demonstrated link to health outcomes [Alternate Mediterranean Diet (aMED) index, Dietary Approach to Stop Hypertension (DASH) score, and Alternative Healthy Eating Index–2010 (AHEI-2010] (15). In addition, we calculated a Paleolithic diet pattern (PALEO) score, which has a different set of emphases and, to our knowledge, has not been investigated in relation to breast cancer risk. We examined each of these indexes in relation to incident pre- and postmenopausal breast cancer risk and hypothesized that each would be inversely associated with the development of breast cancer.

## Methods

### Study population

The California Teachers Study (CTS) is an ongoing prospective cohort study consisting of 133,479 women aged 22–104 y at enrollment who were current and former public school teachers and administrators and who completed a 16-page mailed questionnaire at study entry in 1995–1996 (16). The use of CTS participants' data for this analysis was approved by the institutional review boards at the City of Hope, the University of Southern California, the Cancer Prevention Institute of California (formerly the Northern California Cancer Center), and the University of California at Irvine, and by the Committee for the Protection of Human Subjects, California Health and Human Services Agency.

### Analytic sample

Participants were excluded if they had a self-reported history of diabetes, heart attack, stroke, or a cancer diagnosis before baseline (n = 21,139) or had excessive missing dietary data (defined as missing dietary data for ≥26 food items out of 103) or extreme caloric intake values at <1% or >99% of the population distribution (i.e., <684 kcal/d, >3362 kcal/d) to limit potential confounding that may occur and to improve the validity of energy-adjusted dietary measures (n=15,381), yielding a final analytic cohort consisting of 96,959 female participants (Supplemental Figure 1).

**Dietary assessment and computation of dietary indexes**

At baseline, a 103-item, semiquantitative FFQ assessed usual dietary habits during the preceding year. This instrument was derived from the early version of the Block 95 FFQ and consisted of 8 major dietary categories, and has been validated in this cohort (17). This self-administered questionnaire defined portion sizes and frequency of consumption (i.e., never or <1 time/mo, 1 time/mo, 2–3 times/mo, 1 time/wk, 2 times/wk, 3–4 times/wk, 5–6 times/wk, every day, or ≥2 times/d) for all 103 items. aMED index. The aMED index characterizes an individual's adherence to a Mediterranean-like dietary pattern (18, 19). The aMED was scored +1 if intake amounts were greater than or equal to the study population median intake for fruits, vegetables, nuts and legumes, fish, and whole grains and MUFA-to-SFA ratio (proxy for olive oil intake); it was also scored +1 for intake amounts less than or equal to the median for red and processed meats (Supplemental Table 1). Alcohol intake was scored +1 for light to moderate intake amounts (i.e., >0–15 g/d). The range of the score was 0–8, with higher scores representing higher adherence. Of note, we combined nut and legume categories as previously done to account for the few items assessed and the many participants who reported no intake of either group (19). In addition, we calculated the index as 7 components, excluding alcohol from the index to better account for potential confounding due to its role as a risk factor for breast cancer (8).

**DASH index.**

The DASH dietary index characterizes adherence to a dietary pattern that emphasizes the intake of vegetables, fruits, nuts and legumes, whole grains, and low-fat dairy, and de-emphasizes sodium-rich foods, sweetened beverages, and red and processed meats (20). Each participant was scored 1 to 5 for each of the 8 categories on the basis of their ranking into quintiles of the overall study population intake. Specifically, for each higher quintile of intake of vegetables, fruits, nuts and legumes, whole grains, and low-fat dairy products, participants received a score of 1 to 5 (e.g., participants in highest quintile were scored +5). Categories of sodium-rich foods, sweetened beverages, and red and processed meats were reverse scored by higher quintiles of intake (e.g., participants in highest quintile were scored +1) (Supplemental Table 1). The range of the score was 8–40, with higher scores representing higher adherence.

**AHEI-2010.**

The AHEI was created in 2002 based on foods and nutrients predictive of chronic disease risk (21). The index characterizes adherence to a dietary pattern that emphasizes intakes of vegetables, fruits, nuts and legumes, whole grains, dietary PUFA intake (overall and long-chain n–3 FAs), and no to moderate alcohol intake, and de-emphasizes the intake of trans FAs, red and processed meats, sodium, and sweetened beverages. The AHEI-2010 has traditionally been composed of 11 components, and the score ranged from 0 to 110 points on the basis of ranking the study population into deciles of intake (15, 21). However, we used a modified index as has been done previously due to the lack of trans FAs and long-chain n–3 FAs in the CTS nutrient database (15). Specifically, each participant was scored 1 to 10 for each of the 9 categories on the basis of their ranking into deciles (foods and nutrients) of the overall study population intake. For each higher decile of intake of vegetables, fruits, nuts and legumes, and whole grains and sum of MUFAs and PUFAs to SFAs (substitute for trans and n–3 FAs), participants received a score of 1 to 10 (e.g., participants in the highest decile were scored +10). Alcohol was scored +10 for light to moderate alcohol intake (i.e., >0–22.5 g/d), whereas heavy (>22.5 g/d) and nondrinkers were scored +0 and +2.5, respectively. Categories of sodium-rich foods, sweetened beverages, and red and processed meats were reverse scored by higher deciles of intake (e.g., participants in the highest decile were scored +1). In addition, we calculated the index as 8 components, excluding alcohol from the index to better account for potential confounding due to its role as a risk factor for breast cancer (8). The range of the score was 8–90 with alcohol and 8–80 without alcohol, with higher scores representing higher adherence. Further details are noted in Supplemental Table 1.

**PALEO index.**

The PALEO index characterizes an individual's adherence to emulating a hunter-gatherer's diet of the Paleolithic era. It is hypothesized to be ideal for health from an adaptive perspective (22, 23). The composition of the pattern is informed by anthropological evidence suggesting that dietary intake during this era was largely vegetables, fruits, nuts and seeds, eggs, and meat with little or no grains, legumes, dairy, and alcohol (23). For studying this dietary pattern in the context of the present day, the few studies that have created a PALEO index have utilized this central concept with subtle variations with similar statistical scoring to the aforementioned indexes in this article. The index we created was modeled after these studies with minor adjustments (22, 24). Each participant was scored 1 to 5 for each of the 13 categories on the basis of their ranking into quintiles of the overall study population intake. Specifically, for each higher quintile of intake of vegetables, green leafy vegetables, whole fruits, nuts and seeds, fish, and nonfried and unprocessed meat (eggs, red meat, and poultry), participants received a score of 1 to 5 (e.g., participants in highest quintile were scored +5). Categories of dairy, grains/breads/snacks, sugar-sweetened beverages, sweetened foods, alcohol, legumes, and processed and fried meats were reverse scored by higher quintiles of intake (e.g., participants in highest quintile of intake were scored +1) (Supplemental Table 1). The range of the score was 13–65, with higher scores representing higher adherence.

**Covariate and confounder assessment**

The baseline questionnaire collected self-reported demographic and lifestyle-related characteristics (16)—specifically, participants' race, menopausal status, hormone replacement therapy status, age at menarche, family history of breast cancer, BMI (kg/m2), parity status (parous or nonparous), oral contraceptive use (never or ever use), smoking status (never, former, or current), alcohol intake (grams per day) when not included in the dietary index of interest, and physical activity levels (moderate or strenuous intensity).

Of note, 8500 women reported an unknown menopausal status. Participants who reported an unknown menopausal status and reported having a hysterectomy between ages 45 and 55 y were classified as being postmenopausal (n = 3024). We used multiple imputation approaches to estimate age at menopause for women between the ages of 40 and 60 y who reported an unknown menopausal status (n = 5281) (25). Women aged <40 y or >60 y were assigned to premenopausal and postmenopausal status, respectively (n = 195).

The variable serving as a marker of socioeconomic status (SES) was derived by combining 4 different domains assessed at baseline. Specifically, the composite SES variable incorporated the participants' residential address to US Census–block data, occupation, education, and family income (26).

**Incident breast cancer ascertainment**

All incident cases were identified through linkage with the California Cancer Registry records, a population-based cancer registry for California residents. The state of California reports >99% of all cancer diagnoses to the California Cancer Registry for current residents, so cohort members are actively followed for cancer outcomes without the need for further contact (27). Changes in address were obtained by notifications from participants, annual mailings, and record linkages with multiple sources, including the US Postal Service. Breast cancer was defined by International Classification of Diseases, 10th revision, codes C50.0–50.9.

**Statistical analysis**

Multivariable Cox proportional hazards regression models were utilized to estimate HRs (95% CIs) of invasive breast cancer according to each of the 4 indexes. The diet quality index scores were ranked into quintiles, with the lowest quintile serving as the referent category. Women who were at risk at baseline of postmenopausal breast cancer were followed from the date they completed the baseline questionnaire until diagnosis with invasive breast cancer, death, the end of follow-up (31 December 2011), or loss to follow-up (e.g., moved out of California without further follow-up),whichever occurred first. Women at risk of premenopausal breast cancer were followed from the date they completed the baseline questionnaire until diagnosis of invasive breast cancer during premenopause or until death, end of follow-up, or loss to follow-up during their premenopausal period or until reaching menopause. Overall, 9915 participants were lost to follow-up and there was no evidence that they differed in baseline exposure or confounder status relative to participants who were fully followed up. There was no evidence the proportional hazards assumption was violated through modeling interaction terms of follow-up time with total diet quality score for each index for premenopausal breast cancer or postmenopausal breast cancer. We also tested whether the diet–breast cancer association differed by tumor hormone receptor status via statistical tests for interaction and models stratifying on the outcome. Tests for trend were carried out across the diet score quintiles by modeling a continuous variable assigned to the median value for each diet score quintile. In addition, we conducted sensitivity analyses in which breast cancer cases occurring during the first 2, 3, and 5 y of follow-up were excluded to examine the potential presence of reverse causation in our findings.

**TABLE 1 Baseline characteristics of premenopausal women participants according to quintile of baseline diet quality scores: California Teachers Study[1]**

| Characteristic | Quintile 1 | Quintile 3 | Quintile 5 |
|---|---|---|---|
| aMED index | | | |
| *n* | 6967 | 8781 | 10,636 |
| Age, y | 39 ± 7 | 40 ± 7 | 42 ± 7 |
| Race, % white | 81.2 | 83.8 | 87.3 |
| Smoking status, % ever smoked | 21.8 | 23.9 | 26.1 |
| FHx BrC, % with no first-degree relative with BrC | 87.0 | 87.0 | 86.5 |
| SES, % with high SES | 36.4 | 40.9 | 44.8 |
| BMI, kg/m$^2$ | 24.8 ± 5.5 | 24.4 ± 5.2 | 23.8 ± 4.5 |
| Parity status, % parous | 72.6 | 75.6 | 78.2 |
| OC use, % ever used OCs | 85.1 | 85.7 | 85.6 |
| Age at menarche, y | 6 ± 1 | 6 ± 1 | 5 ± 1 |
| Total energy intake, kcal/d | 1495 + 481 | 1673 ± 526 | 1809 ± 511 |
| Alcohol intake, g ethanol/d | 7.2 ± 10.6 | 7.2 ± 8.7 | 6.4 ± 6.6 |
| Physical activity, h/wk | 1.6 ± 2.1 | 2.1 ± 2.4 | 2.6 ± 2.6 |
| DASH | | | |
| *n* | 7868 | 9759 | 7853 |
| Age, y | 39 ± 8 | 40 ± 7 | 42 ± 7 |
| Race, % white | 76.4 | 85.8 | 89.1 |
| Smoking status, % ever smoked | 21.1 | 24.6 | 27.0 |
| FHx BrC, % with no first-degree relative with BrC | 87.5 | 87.7 | 86.4 |
| SES, % with high SES | 37.3 | 40.7 | 43.5 |
| BMI, kg/m$^2$ | 25.1 ± 5.8 | 24.3 ± 5.0 | 23.5 ± 4.3 |
| Parity status, % parous | 70.6 | 77.0 | 78.4 |
| OC use, % ever used OCs | 84.9 | 86.1 | 84.2 |
| Age at menarche, y | 6 ± 1 | 5 ± 1 | 6 ± 1 |
| Total energy intake, kcal/d | 1652 ± 526 | 1666 ± 526 | 1724 ± 501 |
| Alcohol intake, g ethanol/d | 6.8 ± 9.1 | 7.2 ± 8.7 | 6.3 ± 7.6 |
| Physical activity, h/wk | 1.6 ± 2.1 | 2.1 ± 2.3 | 2.7 ± 2.7 |
| AHEI-2010 | | | |
| *n* | 8413 | 8899 | 8746 |
| Age, y | 38 ± 8 | 41 ± 7 | 43 ± 7 |
| Race, % white | 80.8 | 84.9 | 87.1 |
| Smoking status, % ever smoked | 22.3 | 23.1 | 27.5 |
| FHx BrC, % with no first-degree relative with BrC | 87.4 | 87.5 | 85.9 |
| SES, % with high SES | 35.6 | 42.5 | 45.5 |
| BMI, kg/m$^2$ | 25.1 ± 5.8 | 24.3 ± 5.0 | 23.5 ± 4.3 |
| Parity status, % parous | 71.5 | 77.6 | 77.9 |
| OC use, % ever used OCs | 85.1 | 85.8 | 85.2 |
| Age at menarche, y | 6 ± 1 | 6 ± 1 | 5 ± 1 |
| Total energy intake, kcal/d | 1771 ± 542 | 1665 ± 526 | 1599 ± 483 |
| Alcohol intake, g ethanol/d | 8.1 ± 11.1 | 6.8 ± 8.2 | 5.8 ± 5.4 |
| Physical activity, h/wk | 1.6 ± 2.2 | 2.1 ± 2.4 | 2.6 ± 2.6 |
| PALEO | | | |
| *n* | 9520 | 9056 | 8258 |
| Age, y | 38 ± 7 | 40 ± 7 | 42 ± 7 |
| Race, % white | 82.0 | 84.8 | 85.9 |
| Smoking status, % ever | 21.8 | 24.8 | 26.5 |
| FHx BrC, % with no first-degree relative with BrC | 87.7 | 86.9 | 86.2 |
| SES, % with high SES | 35.4 | 41.6 | 46.3 |
| BMI, kg/m$^2$ | 24.4 ± 5.3 | 24.4 ± 5.1 | 24 ± 4.9 |
| Parity status, % parous | 70.9 | 76.4 | 78.4 |

| Characteristic | Quintile 1 | Quintile 3 | Quintile 5 |
|---|---|---|---|
| OC use, % ever used OCs | 85.8 | 85.6 | 84.2 |
| Age at menarche, y | 6 ± 1 | 6 ± 1 | 5 ± 1 |
| Total energy intake, kcal/d | 1833 ± 525 | 1670 ± 527 | 1498 ± 455 |
| Alcohol intake, g ethanol/d | 8.3 ± 9.1 | 6.9 ± 8.1 | 5.4 ± 7.9 |
| Physical activity, h/wk | 1.8 ± 2.2 | 2.0 ± 2.3 | 2.5 ± 2.7 |

[1]Values are frequencies or means ± SDs. AHEI-2010, Alternative Healthy Eating Index–2010; aMED, Alternate Mediterranean Diet; BrC, breast cancer; DASH, Dietary Approaches to Stop Hypertension; FHx, family history; OC, oral contraceptive; PALEO, Paleolithic index; SES, socioeconomic status.

All multivariable Cox proportional hazards regression models were adjusted for the following covariates at baseline: age, race (white, black, Hispanic, Native American, Asian/Pacific Islander, or unknown), hormone replacement therapy status (no hormone therapy usage, past hormone therapy usage, current estrogen therapy usage, current estrogen and progesterone usage, or unknown), family history of breast cancer (no first-degree relative, ≥1 first-degree relative, adopted, or unknown), age at menarche (in years), parity status (nonparous or parous), oral contraceptive use (never or ever use), smoking status (never, former, or current), SES, physical activity (hours per week; moderate or strenuous), total energy intake (kilocalories per day), total alcohol intake (grams of ethanol per day), when applicable, and BMI (kg/m2). The first Cox regression model (model 1) included all the above-mentioned covariates except for BMI. Model 2 additionally adjusted for BMI, which may be in the causal pathway between diet and the development of breast cancer (28). Pearson's correlation coefficients were calculated among the 4 indexes. All statistical tests were 2-sided and conducted with SAS software (version 9.3; SAS Institute).

**Results**

The study analytic sample of 96,959 participants were aged 52 ± 14 y, on average, and were followed for 1,354,947 person-years; 4826 developed invasive breast cancer during follow-up. Baseline characteristics and HRs are presented separately for premenopausal breast cancer risk and postmenopausal breast cancer risk because there was evidence that the association differed. The scoring details for each dietary pattern are presented in Supplemental Table 1. Dietary characteristics according to the 4 dietary pattern scores are reported in Supplemental Tables 2 and 3, and the correlations among the 4 indexes are presented in Supplemental Table 4.

Of the 96,959 participants, 42,517 were premenopausal at baseline and 346 developed invasive breast cancer during the premenopausal period with 292,943 y of follow-up. Table 1 reports baseline characteristics for participants at risk of premenopausal breast cancer according to quintiles of the dietary pattern scores. In the 54,442 participants who were at risk of postmenopausal breast cancer at baseline, 3523 developed the cancer during 728,233 follow-up y. Table 2 reports baseline characteristics for participants at risk of postmenopausal breast cancer according to quintiles of the dietary pattern scores.

Table 3 shows HRs for the incidence of premenopausal breast cancer according to quintiles of the diet quality scores. Overall, there was no association between any of the scores and incident premenopausal breast cancer. Table 4 shows HRs for the incidence of postmenopausal breast cancer according to quintiles of the diet quality scores. There were similar modest inverse associations between higher aMED, DASH, and AHEI-2010 scores with the risk of developing postmenopausal breast cancer. However, there was no association between PALEO scores and risk of breast cancer. Adjustment for BMI did not materially change the estimates, although it tended to expand the upper 95% CI across 1.0.

Analyses that tested whether the associations between the scores and pre- and postmenopausal breast cancer differed by the tumor hormone receptor status of the cases provided no evidence that associations differed, because the P value for the statistical interaction was >0.88 across all diet scores and stratified analyses did not provide any evidence that the results differed (results not shown). Further analyses that excluded cases that occurred with the first 2, 3, and 5 y of follow-up did not materially alter any of the findings.

**Discussion**

In a population of women in the CTS, we observed a modest inverse association between higher scores on 3 different diet indexes that emphasize higher intakes of whole grains, vegetables and fruits, legumes, and nuts and seeds, and de-emphasize red and processed meats and sugar-sweetened beverages and the risk of incident postmenopausal breast cancer; adjustment for BMI did not materially affect the association but it tended to expand the upper 95% CI bounds across 1.0, precluding strong inference from these estimates. We observed no association between a PALEO score and the risk of postmenopausal breast cancer, and no association between any diet score and premenopausal breast cancer risk.

These results for the diet–postmenopausal breast cancer risk analyses in this study align with previous research that examined the diet–postmenopausal breast cancer risk association. In the Nurses' Health Study (NHS), higher DASH and AHEI diet quality scores were associated with lower postmenopausal breast cancer risk (18, 20). In addition, higher AHEI scores were inversely associated with postmenopausal breast cancer risk among women in a family-based Canadian cohort study (29). Higher aMED index scores have also been associated with a lower risk of postmenopausal breast cancer in previous cohort and case-control studies (30–33). Furthermore, evidence from the Prevención con Dieta Mediterránea (PREDIMED) trial showed that postmenopausal female participants randomly assigned to consume a Mediterranean diet reduced their risk of developing invasive breast cancer when compared with controls (HR: 0.43; 95% CI: 0.21, 0.88) (34). Overall, these results tend to align with the conclusion of current systematic reviews and meta-analyses on the topic (9).

The results for the diet–premenopausal breast cancer risk analyses in this study are also largely inconsistent with the minimal evidence base on the topic. Three prospective cohort studies found no evidence for the association between higher aMED, DASH, and AHEI scores and premenopausal breast cancer risk (30, 35, 36). Notably, the Swedish Women's Lifestyle and Health cohort study found higher aMED scores to be associated with an ∼2-fold increase in the risk of premenopausal breast cancer (37). In addition, related research that used a posteriori–defined dietary pattern analysis methods to examine diet–premenopausal breast cancer risk associations have generally observed that higher scores on patterns with higher intakes of whole grains, vegetables and fruits, legumes, and nuts and seeds and lower intakes of red and processed meats and sugar-sweetened beverages are not associated with premenopausal breast cancer (4, 11, 35, 38, 39). Three of the 4 diet quality scores (AHEI-2010, DASH, and aMED) included in

**TABLE 2 Baseline characteristics of postmenopausal women participants according to quintile of baseline diet quality scores: California Teachers Study[1]**

| Characteristic | Quintile 1 | Quintile 3 | Quintile 5 |
|---|---|---|---|
| aMED index | | | |
| *n* | 10,413 | 11,649 | 11,926 |
| Age, y | 59 ± 10 | 61 ± 10 | 62 ± 10 |
| Race, % white | 89.6 | 90.7 | 90.7 |
| HRT status, % never used HRT | 19.8 | 19.6 | 19 |
| Smoking status, % ever smoked | 45.6 | 48.0 | 51.3 |
| FHx BrC, % with no first-degree relative with BrC | 83.8 | 83.9 | 84.0 |
| SES, % with high SES | 45.6 | 48.0 | 51.3 |
| BMI, kg/m$^2$ | 81.4 | 83.2 | 84.2 |
| Parity status, % parous | 64.1 | 58.9 | 55.2 |
| OC use, % ever used OCs | 6 ± 1 | 6 ± 1 | 6 ± 1 |
| Age at menarche, y | 25.5 ± 5.3 | 25.2 ± 4.8 | 24.6 ± 4.4 |
| Total energy intake, kcal/d | 1372 ± 443 | 1518 ± 485 | 1662 ± 472 |
| Alcohol intake, g ethanol/d | 9.9 ± 12.4 | 8.5 ± 10.2 | 6.8 ± 7.4 |
| Physical activity, h/wk | 2.0 ± 2.2 | 2.3 ± 2.4 | 2.7 ± 2.5 |
| DASH | | | |
| *n* | 9738 | 12,606 | 10,380 |
| Age, y | 57 ± 9 | 61 ± 10 | 65 ± 10 |
| Race, % white | 85.8 | 90.7 | 92.7 |
| HRT status, % never used HRT | 19.7 | 18.6 | 19.6 |
| Smoking status, % ever smoked | 42.5 | 40.6 | 38.2 |
| FHx BrC, % with no first-degree relative with BrC | 84.3 | 83.7 | 83.7 |
| SES, % with high SES | 46.6 | 48.2 | 49.7 |
| BMI, kg/m$^2$ | 25.8 ± 5.5 | 25.1 ± 4.8 | 24.2 ± 4.1 |
| Parity status, % parous | 81.4 | 83.4 | 83.8 |
| OC use, % ever used OCs | 68.7 | 60.0 | 49.4 |
| Age at menarche, y | 6 ± 1 | 6 ± 1 | 6 ± 1 |
| Total energy intake, kcal/d | 1510 ± 496 | 1520 ± 491 | 1553 ± 447 |
| Alcohol intake, g ethanol/d | 9.1 ± 11.4 | 8.5 ± 10.0 | 6.9 ± 8.7 |
| Physical activity, h/wk | 2.0 ± 2.2 | 2.3 ± 2.3 | 2.7 ± 2.6 |
| AHEI-2010 | | | |
| *n* | 10,815 | 10,217 | 10,941 |
| Age, y | 58 ± 10 | 61 ± 10 | 64 ± 10 |
| Race, % white | 89.1 | 90.0 | 90.9 |
| HRT status, % never used HRT | 19.1 | 19.2 | 20.2 |
| Smoking status, % ever smoked | 44.4 | 38.7 | 38.5 |
| FHx BrC, % with no first-degree relative with BrC | 83.8 | 83.2 | 83.8 |
| SES, % with high SES | 45.7 | 47.3 | 51.4 |
| BMI, kg/m$^2$ | 25.9 ± 5.5 | 25.1 ± 4.7 | 24.1 ± 4.0 |
| Parity status, % parous | 80.7 | 83.8 | 83.6 |
| OC use, % ever used OCs | 67.0 | 59.7 | 52.2 |
| Age at menarche, y | 6 ± 1 | 6 ± 1 | 6 ± 1 |
| Total energy intake, kcal/d | 1618 ± 514 | 1504 ± 485 | 1470 ± 440 |
| Alcohol intake, g ethanol/d | 11.4 ± 13.5 | 7.6 ± 9.6 | 5.8 ± 5.7 |
| Physical activity, h/wk | 1.9 ± 2.5 | 2.4 ± 2.7 | 3.2 ± 3.1 |
| PALEO | | | |
| *n* | 10,562 | 11,976 | 11,516 |
| Age, y | 59 ± 10 | 61 ± 10 | 62 ± 10 |
| Race, % white | 89.1 | 90.7 | 90.5 |
| HRT status, % never used HRT | 18.1 | 18.8 | 21 |
| Smoking status, % ever smoked | 42.4 | 40.7 | 39.4 |

| Characteristic | Quintile | | |
| --- | --- | --- | --- |
| | 1 | 3 | 5 |
| FHx BrC, % with no first-degree relative with BrC | 84.2 | 83.2 | 84.0 |
| SES, % with high SES | 43.8 | 48.2 | 52.3 |
| BMI, kg/m$^2$ | 25.3 ± 5.1 | 25.0 ± 4.6 | 24.8 ± 4.5 |
| Parity status, % parous | 81.9 | 83.4 | 83.4 |
| OC use, % ever used OCs | 65.5 | 59.5 | 54.5 |
| Age at menarche, y | 6 ± 1 | 6 ± 1 | 5 ± 1 |
| Total energy intake, kcal/d | 1651 ± 512 | 1530 ± 491 | 1381 ± 402 |
| Alcohol intake, g ethanol/d | 9.7 ± 10.7 | 8.6 ± 10.3 | 6.5 ± 9.2 |
| Physical activity, h/wk | 2.2 ± 2.3 | 2.3 ± 2.4 | 2.5 ± 2.5 |

[1]Values are frequencies or means ± SDs. AHEI-2010, Alternative Healthy Eating Index–2010; aMED, Alternate Mediterranean Diet; BrC, breast cancer; DASH, Dietary Approaches to Stop Hypertension; FHx, family history; HRT, hormone replacement therapy; OC, oral contraceptive; PALEO, Paleolithic index; SES, socioeconomic status.

**TABLE 3 HRs (95% CIs) of invasive premenopausal breast cancer according to diet quality scores in women: California Teachers Study[1]**

| Diet index, quintile (score range) | Cases, *n* | Participants, *n* | HR (95% CI) Model 1[2] | HR (95% CI) Model 2[3] |
|---|---|---|---|---|
| aMED | | | | |
| Q1 (0–2) | 59 | 6967 | 1.00 (Ref) | 1.00 (Ref) |
| Q2 (3) | 55 | 7264 | 1.06 (0.86, 1.29) | 1.06 (0.86, 1.29) |
| Q3 (4) | 72 | 8781 | 1.10 (0.91, 1.33) | 1.10 (0.91, 1.33) |
| Q4 (5) | 59 | 8869 | 1.16 (0.96, 1.40) | 1.15 (0.96, 1.39) |
| Q5 (6–8) | 101 | 10,636 | 1.15 (0.95, 1.38) | 1.14 (0.95, 1.38) |
| aMED[4] | | | | |
| Q1 (0–2) | 82 | 9209 | 1.00 (Ref) | 1.00 (Ref) |
| Q2 (3) | 58 | 8136 | 0.95 (0.79, 1.14) | 0.95 (0.79, 1.14) |
| Q3 (4) | 69 | 9301 | 0.98 (0.82, 1.16) | 0.98 (0.82, 1.16) |
| Q4 (5) | 66 | 8601 | 1.12 (0.95, 1.33) | 1.12 (0.94, 1.33) |
| Q5 (6–7) | 71 | 7270 | 1.04 (0.87, 1.25) | 1.04 (0.87, 1.25) |
| DASH | | | | |
| Q1 (8–19) | 69 | 7868 | 1.00 (Ref) | 1.00 (Ref) |
| Q2 (20–22) | 69 | 8647 | 1.01 (0.84, 1.21) | 1.01 (0.84, 1.21) |
| Q3 (23–25) | 76 | 9759 | 1.02 (0.86, 1.22) | 1.02 (0.86, 1.22) |
| Q4 (26–28) | 61 | 8390 | 0.89 (0.74, 1.08) | 0.89 (0.74, 1.07) |
| Q5 (29–40) | 71 | 7853 | 1.03 (0.86, 1.24) | 1.03 (0.86, 1.23) |
| AHEI-2010 | | | | |
| Q1 (8–41) | 76 | 8413 | 1.00 (Ref) | 1.00 (Ref) |
| Q2 (41.5–47) | 62 | 8626 | 0.84 (0.70, 1.01) | 0.84 (0.70, 1.01) |
| Q3 (47.5–52.5) | 60 | 8899 | 1.02 (0.85, 1.21) | 1.02 (0.85, 1.21) |
| Q4 (53–59.5) | 71 | 7833 | 1.06 (0.89, 1.27) | 1.06 (0.89, 1.27) |
| Q5 (60–90) | 77 | 8746 | 0.95 (0.80, 1.13) | 0.95 (0.79, 1.13) |
| AHEI-2010[4] | | | | |
| Q1 (8–35) | 77 | 8573 | 1.00 (Ref) | 1.00 (Ref) |
| Q2 (36–41) | 68 | 8759 | 1.07 (0.90, 1.29) | 1.07 (0.90, 1.29) |
| Q3 (42–46) | 57 | 8125 | 0.99 (0.82, 1.19) | 0.99 (0.82, 1.19) |
| Q4 (47–52) | 65 | 8347 | 1.04 (0.87, 1.25) | 1.04 (0.87, 1.25) |
| Q5 (53–80) | 79 | 8713 | 1.08 (0.90, 1.29) | 1.08 (0.90, 1.29) |
| PALEO | | | | |
| Q1 (13–34) | 80 | 9520 | 1.00 (Ref) | 1.00 (Ref) |
| Q2 (35–37) | 66 | 8183 | 1.01 (0.84, 1.21) | 1.01 (0.84, 1.21) |
| Q3 (38–40) | 79 | 9056 | 1.02 (0.85, 1.21) | 1.02 (0.85, 1.21) |
| Q4 (41–43) | 55 | 7500 | 1.02 (0.85, 1.22) | 1.01 (0.85, 1.22) |
| Q5 (44–65) | 66 | 8258 | 1.10 (0.92, 1.31) | 1.10 (0.92, 1.31) |

1n = 42,517. AHEI-2010, Alternative Healthy Eating Index–2010; aMED, Alternate Mediterranean Diet; DASH, Dietary Approaches to Stop Hypertension; PALEO, Paleolithic index; Q, quintile; Ref, reference.

[2]Adjusted for age at baseline, race, breast cancer family history, age at menarche, oral contraceptive use, parity status, smoking status, socioeconomic status, physical activity, total energy intake (kilocalories per day), and total alcohol intake (grams of ethanol per day) (for dietary indexes without alcohol intake).

[3]Adjusted as for model 1 and also adjusted for BMI at baseline.

[4]Dietary index formulated without the inclusion of alcohol intake and alcohol included as covariate (total alcohol; grams of ethanol per day) in the multivariable models.

**TABLE 4** HRs (95% CIs) of invasive postmenopausal breast cancer according to diet quality scores in women: California Teachers Study[1]

| Diet index, quintile (score range) | Cases, *n* | Participants, *n* | Model 1[2] | | Model 2[3] | |
|---|---|---|---|---|---|---|
| | | | HR (95% CI) | *P*-trend | HR (95% CI) | *P*-trend |
| aMED | | | | | | |
| Q1 (0–2) | 670 | 10,413 | 1.00 (Ref) | | 1.00 (Ref) | |
| Q2 (3) | 646 | 9755 | 0.99 (0.89, 1.10) | | 0.99 (0.89, 1.11) | |
| Q3 (4) | 754 | 11,649 | 0.94 (0.85, 1.05) | | 0.95 (0.85, 1.05) | |
| Q4 (5) | 675 | 10,699 | 0.90 (0.81, 1.01) | | 0.91 (0.82, 1.02) | |
| Q5 (6–8) | 778 | 11,926 | 0.91 (0.82, 1.02) | 0.03 | 0.93 (0.83, 1.03) | 0.06 |
| aMED[4] | | | | | | |
| Q1 (0–2) | 849 | 13,159 | 1.00 (Ref) | | 1.00 (Ref) | |
| Q2 (3) | 696 | 10,856 | 0.95 (0.86, 1.06) | | 0.96 (0.87, 1.06) | |
| Q3 (4) | 782 | 12,034 | 0.95 (0.86, 1.05) | | 0.96 (0.87, 1.06) | |
| Q4 (5) | 666 | 10,288 | 0.94 (0.84, 1.04) | | 0.95 (0.85, 1.05) | |
| Q5 (6–7) | 530 | 8105 | 0.93 (0.83, 1.04) | 0.18 | 0.95 (0.85, 1.06) | 0.33 |
| DASH | | | | | | |
| Q1 (8–19) | 649 | 9738 | 1.00 (Ref) | | 1.00 (Ref) | |
| Q2 (20–22) | 701 | 10,789 | 0.93 (0.84, 1.04) | | 0.94 (0.84, 1.04) | |
| Q3 (23–25) | 818 | 12,606 | 0.92 (0.83, 1.02) | | 0.92 (0.83, 1.03) | |
| Q4 (26–28) | 685 | 10,929 | 0.88 (0.78, 0.98) | | 0.89 (0.79, 0.99) | |
| Q5 (29–40) | 670 | 10,380 | 0.89 (0.80, 1.00) | 0.03 | 0.91 (0.81, 1.02) | 0.07 |
| AHEI-2010 | | | | | | |
| Q1 (8–41) | 728 | 10,815 | 1.00 (Ref) | | 1.00 (Ref) | |
| Q2 (41.5–47) | 731 | 11,254 | 0.93 (0.84, 1.03) | | 0.93 (0.84, 1.03) | |
| Q3 (47.5–52.5) | 662 | 10,217 | 0.92 (0.83, 1.02) | | 0.93 (0.83, 1.03) | |
| Q4 (53–59.5) | 704 | 11,215 | 0.86 (0.78, 0.96) | | 0.87 (0.78, 0.97) | |
| Q5 (60–90) | 698 | 10,941 | 0.87 (0.78, 0.97) | 0.004 | 0.88 (0.79, 0.98) | 0.01 |
| AHEI-2010[4] | | | | | | |
| Q1 (8–35) | 690 | 10,706 | 1.00 (Ref) | | 1.00 (Ref) | |
| Q2 (36–41) | 766 | 11,248 | 1.01 (0.91, 1.12) | | 1.02 (0.92, 1.13) | |
| Q3 (42–46) | 685 | 10,853 | 0.91 (0.82, 1.01) | | 0.92 (0.83, 1.03) | |
| Q4 (47–52) | 716 | 11,144 | 0.92 (0.83, 1.03) | | 0.94 (0.84, 1.04) | |
| Q5 (53–80) | 666 | 10,491 | 0.90 (0.81, 1.01) | 0.02 | 0.92 (0.82, 1.03) | 0.05 |
| PALEO | | | | | | |
| Q1 (13–34) | 626 | 10,562 | 1.00 (Ref) | | 1.00 (Ref) | |
| Q2 (35–37) | 660 | 10,190 | 1.06 (0.95, 1.18) | | 1.06 (0.95, 1.18) | |
| Q3 (38–40) | 825 | 11,976 | 1.11 (1.00, 1.23) | | 1.11 (1.00, 1.23) | |
| Q4 (41–43) | 659 | 10,198 | 1.03 (0.92, 1.15) | | 1.03 (0.93, 1.16) | |
| Q5 (44–65) | 753 | 11,516 | 1.05 (0.94, 1.17) | 0.61 | 1.05 (0.94, 1.17) | 0.56 |

[1]n = 54,442. AHEI-2010, Alternative Healthy Eating Index–2010; aMED, Alternate Mediterranean Diet; DASH, Dietary Approaches to Stop Hypertension; PALEO, Paleolithic index; Q, quintile; Ref, reference.
[2]Adjusted for age at baseline, race, breast cancer family history, age at menarche, oral contraceptive use, parity status, smoking status, socioeconomic status, physical activity, total energy intake (kilocalories per day), and total alcohol intake (grams of ethanol per day) (for dietary indexes without alcohol intake).
[3]Adjusted as for model 1 and also adjusted for BMI at baseline.
[4]Dietary index formulated without the inclusion of alcohol intake and alcohol included as a covariate (total alcohol; grams of ethanol per day) in the multivariable models.

this study showed modest inverse associations with breast cancer risk in postmenopausal women. These 3 diet scores generally overlap in their recommendations for various food groups, such as higher intakes of whole grains, fruits and vegetables, legumes, and nuts and seeds and lower intakes of sweetened beverages and red and processed meats. Our results suggest that higher intakes of similar components among these diet quality scores (aMED, DASH, and AHEI-2010) may have a preventive role in postmenopausal breast cancer.

The examination of the PALEO index with breast cancer risk was a novel aspect of this study, albeit there was no association with breast cancer. However, these null results may provide insight into differences in associations from the tested diet quality scores. The PALEO index emphasizes intakes of vegetables, green leafy vegetables, whole fruits, nuts and seeds, fish, eggs, and nonfried and unprocessed meat (red meat and poultry) and de-emphasizes processed and fried meats, cereals, sweet foods, breads, snacks and grains, and alcohol. Higher whole-grain and legume intake is not emphasized in the PALEO index as in the comparative indexes and may explain some of the lack of association between the PALEO index and breast cancer risk, because previous research has shown a strong inverse association between consistent consumption of whole grains and the risk of breast cancer by 51% (OR for >7 times/wk compared with never or rarely: 0.49; 95% CI: 0.29, 0.82) (40). Furthermore, higher whole-grain intake has a large body of evidence both from observational and experimental studies supporting beneficial effects on pathways such as insulin resistance, oxidative stress, and inflammation, which are thought to contribute to breast cancer carcinogenesis (41, 42). In addition, higher red meat intake has been associated with an increased risk of breast cancer (43), and this dietary element is emphasized in the PALEO pattern score. There are several strengths of this study. It is a prospective study with a long period of follow-up. The large sample size and number of cases provided ample statistical power to investigate diet and incident breast cancer risk (16). The dietary data collected have been shown to be reliable and valid (17), and the use of dietary pattern scores to study the diet–breast cancer relation may be more effectively translated into population-based recommendations. We also had the ability to examine breast cancer by hormone receptor status, although overall diet quality was not associated with the risk of breast cancer subtype groups, a finding consistent with previous dietary analyses from the cohort (2).

Limitations include some degree of measurement error with the dietary assessment, although this likely represents nondifferential misclassification with respect to disease status, pushing estimates toward the null. We also had to exclude ∼15% of the study population due to inadequate dietary data for analysis, and another 10.2% of the analytic population were lost to follow-up, although they did not differ in any material way from the analytic population with ascertained cancer status. The self-report of other lifestyle-related data may also result in misclassification and residual confounding in our models. In addition, changes in BMI and diet occurring after baseline would further inform the study results but were not available.

In conclusion, diet quality indexes that emphasize higher intakes of whole grains, vegetables and fruits, legumes, and nuts and seeds and de-emphasize red and processed meats and sweetened foods were modestly associated with a lower risk of incident postmenopausal breast cancer, and there was no association between the PALEO diet score and the risk of postmenopausal breast cancer, and no association between any diet score and premenopausal breast cancer risk.

**Acknowledgments**

The CTS steering committee includes Leslie Bernstein, Susan Neuhausen, James Lacey, Sophia Wang, Huiyan Ma, and Jessica Clague DeHart at the Beckman Research Institute of City of Hope; Dennis Deapen, Rich Pinder, and Eunjung Lee at the University of Southern California; Hoda Anton-Culver, Argyrios Ziogas, and Hannah Park at the University of California Irvine; and Fred Schumaker at Case Western University.
The authors' responsibilities were as follows—VH and AOO: designed the specific research project and take primary responsibility for the final content of the manuscript; AZ, SLN, and HA-C: contributed to the formation and maintenance of the cohort, including the data collection; VH, AZ, and AOO: performed the data analysis; VH, and AOO: wrote the manuscript; and all authors: made intellectual contributions to and read and approved the final manuscript.

**References**

1. DeSantis C. Breast cancer statistics, 2013. CA Cancer J Clin 2014;64:52–62.
2. Link LB, Canchola AJ, Bernstein L, Clarke CA, Stram DO, Ursin G, Horn-Ross PL. Dietary patterns and breast cancer risk in the California Teachers Study cohort. Am J Clin Nutr 2013;98:1524–32.
3. Hirko KA, Willett WC, Hankinson SE, Rosner BA, Beck AH, Tamimi RM, Eliassen AH. Healthy dietary patterns and risk of breast cancer by molecular subtype. Breast Cancer Res Treat 2016;155:579–88.
4. Karimi Z, Jessri M, Houshiar-Rad A, Mirzaei HR, Rashidkhani B. Dietary patterns and breast cancer risk among women. Public Health Nutr 2014;17:1098–106.
5. Tabung FK, Steck SE, Liese AD, Zhang J, Ma Y, Johnson KC, Lane DS, Qi L, Snetselaar L, Vitolins MZ, et al. Patterns of change over time and history of the inflammatory potential of diet and risk of breast cancer among postmenopausal women. Breast Cancer Res Treat 2016;159(1):139–49.
6. Jordan I, Hebestreit A, Swai B, Krawinkel MB. Dietary patterns and breast cancer risk among women in northern Tanzania: a case-control study. Eur J Nutr 2013;52:905–15.
7. Baglietto L, Krishnan K, Severi G, Hodge A, Brinkman M, English DR, McLean C, Hopper JL, Giles GG. Dietary patterns and risk of breast cancer. Br J Cancer 2011;104:524–31.
8. Albuquerque RC, Baltar VT, Marchioni DM. Breast cancer and dietary patterns: a systematic review. Nutr Rev 2014;72:1–17.
9. Brennan SF, Cantwell MM, Cardwell CR, Velentzis LS, Woodside JV. Dietary patterns and breast cancer risk: a systematic review and metaanalysis. Am J Clin Nutr 2010;91:1294–302.
10. Bahadoran Z, Zeinab K, Somayeh A. Healthy dietary patterns and the risk of breast cancer: a review of current data. Am J Life Sci 2014;3:
11. Castelló A, Pollán M, Buijsse B, Ruiz A, Casas AM, Baena-Cañada JM, Lope V, Antolín S, Ramos M, Muñoz M, et al. Spanish Mediterranean diet and other dietary patterns and breast cancer risk: case-control EpiGEICAM study. Br J Cancer 2014;111: 1454–62.
12. McGuire S. Scientific report of the 2015 Dietary Guidelines Advisory Committee. Adv Nutr 2016;7:202–4.
13. Thomson CA. Diet and breast cancer understanding risks and benefits. Nutr Clin Pract 2012;27:636–50.

14. Patterson RE, Cadmus LA, Emond JA, Pierce JP. Physical activity, diet, adiposity and female breast cancer prognosis: a review of the epidemiologic literature. Maturitas 2010;66: 5–15.
15. Harmon BE, Boushey CJ, Shvetsov YB, Ettienne R, Reedy J, Wilkens LR, Le Marchand L, Henderson BE, Kolonel LN. Associations of key diet-quality indexes with mortality in the Multiethnic cohort: the
Dietary Patterns Methods Project. Am J Clin Nutr 2015;101:587–97.
16. Bernstein L, Allen M, Anton-Culver H, Deapen D, Horn-Ross PL, Peel D, Pinder R, Reynolds P, Sullivan-Halley J, West D, et al. High breast cancer incidence rates among California teachers: results from the California Teachers Study (United States). Cancer Causes Control 2002;13:625–35.
17. Horn-Ross PL, Lee VS, Collins CN, Stewart SL, Canchola AJ, Lee MM, Reynolds P, Clarke CA, Bernstein L, Stram DO. Dietary assessment in the California Teachers Study: reproducibility and validity. Cancer Causes Control 2008;19:595–603.
18. Fung TT, Hu FB, McCullough ML, Newby PK, Willett WC, Holmes MD. Diet quality is associated with the risk of estrogen receptor-negative breast cancer in postmenopausal women. J Nutr 2006;136:466–72.
19. Schwingshackl L, Hoffmann G. Adherence to Mediterranean diet and risk of cancer: a systematic review and meta-analysis of observational studies. Int J Cancer 2014;135:1884–97.
20. Fung TT, Hu FB, Hankinson SE, Willett WC, Holmes MD. Low-carbohydrate diets, dietary approaches to stop hypertension-style diets, and the risk of postmenopausal breast cancer. Am J Epidemiol 2011;174:652–60.
21. Chiuve SE, Fung TT, Rimm EB, Hu FB, McCullough ML, Wang M, Stampfer MJ, Willett WC. Alternative dietary indices both strongly predict risk of chronic disease. J Nutr 2012;142:1009–18.
22. Whalen KA, McCullough ML, Flanders WD, Hartman TJ, Judd S, Bostick RM. Paleolithic and Mediterranean diet pattern scores are inversely associated with biomarkers of inflammation and oxidative balance in adults. J Nutr 2016;146:1217–26.
23. Katz DL, Meller S. Can we say what diet is best for health? Annu Rev Public Health 2014;18:83–103
24. Whalen KA, McCullough M, Flanders WD, Hartman TJ, Judd S, Bostick RM. Paleolithic and Mediterranean diet pattern scores and risk of incident, sporadic colorectal adenomas. Am J Epidemiol 2014;180:1088–97.
25. Rosner B, Colditz GA. Age at menopause: imputing age at menopause for women with a hysterectomy with application to risk of postmenopausal breast cancer. Ann Epidemiol 2011;21:450–60.
26. Reynolds P, Hurley S, Goldberg DE, Anton-Culver H, Bernstein L, Deapen D, Horn-Ross PL, Peel D, Pinder R, Ross RK, et al. Regional variations in breast cancer among California teachers. Epidemiology 2004;15:746–54.
27. Kwong SL, Perkins CI,Morris CR, Cohen R, AllenM, Schlag R, Wright WE. Cancer in California: 1988–1999. Sacramento, CA: California Department of Health Services, Cancer Surveillance Section; 2001.
28. Cheraghi Z, Poorolajal J, Hashem T, Esmailnasab N, Irani AD. Effect of body mass index on breast cancer during premenopausal and postmenopausal periods: a meta-analysis. PloS One 2012;7:e51446.

29. Nkondjock A, Ghadirian P. Diet quality and BRCA-associated breast cancer risk. Breast Cancer Res Treat 2007;103:361–9.
30. Buckland G, Travier N, Cottet V, Gonzalez CA, Luján-Barroso L, Agudo A, Trichopoulou A, Lagiou P, Trichopoulos D, Peeters PH, et al. Adherence to the Mediterranean diet and risk of breast cancer in the European Prospective Investigation into Cancer and Nutrition cohort study. Int J Cancer 2013;132:2918–27.
31. Trichopoulou A, Bamia C, Lagiou P, Trichopoulos D. Conformity to traditional Mediterranean diet and breast cancer risk in the Greek EPIC (European Prospective Investigation into Cancer and Nutrition) cohort. Am J Clin Nutr 2010;92:620–5.
32. Wu AH, Yu MC, Tseng CC, Stanczyk FZ, Pike MC. Dietary patterns and breast cancer risk in Asian American women. Am J Clin Nutr 2009;89:1145–54.
33. van den Brandt PA, Schulpen M. Mediterranean diet adherence and risk of postmenopausal breast cancer: results of a cohort study and meta-analysis. Int J Cancer 2017;140:2220–31.
34. Toledo E, Salas-Salvadó J, Donat-Vargas C, Buil-Cosiales P, Estruch R, Ros E, Corella D, Fitó M, Hu FB, Arós F, et al. Mediterranean diet and invasive breast cancer risk among women at high cardiovascular risk in the PREDIMED trial: a randomized clinical trial. JAMA Intern Med 2015;175:1752–60.
35. Harris HR, Willett WC, Vaidya RL, Michels KB. Adolescent dietary patterns and premenopausal breast cancer incidence. Carcinogenesis 2016;37:376–84.
36. Nilsson LM, Winkvist A, Johansson I, Lindahl B, Hallmans G, Lenner P, Van Guelpen B. Low-carbohydrate, high-protein diet score and risk of incident cancer; a prospective cohort study. Nutr J 2013;12:58.
37. Couto E, Sandin S, Löf M, Ursin G, Adami HO, Weiderpass E. Mediterranean dietary pattern and risk of breast cancer. PloS One 2013;8:e55374.
38. Baglietto L, Krishnan K, Severi G, Hodge A, Brinkman M, English DR, McLean C, Hopper JL, Giles GG. Dietary patterns and risk of breast cancer. Br J Cancer 2011;104:524.
39. Catsburg C, Kim RS, Kirsh VA, Soskolne CL, Kreiger N, Rohan TE. Dietary patterns and breast cancer risk: a study in 2 cohorts. Am J Clin Nutr 2015;101:817–23.
40. Mourouti N, Kontogianni MD, Papavagelis C, Psaltopoulou T, Kapetanstrataki MG, Plytzanopoulou P, Vassilakou T, Malamos N, Linos A, Panagiotakos DB.Whole grain consumption and breast cancer: a case-control study in women. J Am Coll Nutr 2016;35:143–9.
41. Ye EQ, Chacko SA, Chou EL, Kugizaki M, Liu S. Greater whole-grain intake is associated with lower risk of type 2 diabetes, cardiovascular disease, and weight gain. J Nutr 2012;142:1304–13.
42. Montonen J, Boeing H, Fritsche A, Schleicher E, Joost HG, Schulze MB, Steffen A, Pischon T. Consumption of red meat and whole-grain bread in relation to biomarkers of obesity, inflammation, glucose metabolism and oxidative stress. Eur J Nutr 2013;52:337–45.
43. Alexander DD, Morimoto LM, Mink PJ, Cushing CA. A review and meta-analysis of red and processed meat consumption and breast cancer. Nutr Res Rev 2010;23:349–65.

Supported in part by grants R01 CA77398 from the National Cancer Institute (NCI), and contract 97-10500 from the California Breast Cancer Research Fund. The collection of data on cancer incidence used in this study was supported by the California Department of Public Health as part of the statewide cancer-reporting program mandated by California Health and Safety Code Section 103885; the NCI's Surveillance, Epidemiology, and End Results Program under contract HHSN261201000036C awarded to the Cancer Prevention Institute of California; contract HHSN261201000035C awarded to the University of Southern California; contract HHSN261201000034C awarded to the Public Health Institute; and the CDC's National Program of Cancer Registries, under agreement 1U58 DP000807-01 awarded to the Public Health Institute.
The ideas and opinions expressed herein are those of the authors, and endorsement by the California Department of Public Health, NCI, and CDC or their contractors and subcontractors is not intended nor should be inferred. The funding sources did not contribute to the design or conduct of the study or to the writing or submission of the manuscript.
Author disclosures: VH, AZ, SLN, HA-C, and AOO, no conflicts of interest. Supplemental Figure 1 and Supplemental Tables 1–4 are available from the "Supplementary data" link in the online posting of the article and from the same link in the online table of contents at https://academic.oup.com/jn/.
Address correspondence to AOO (e-mail: aodegaar@uci.edu).
Abbreviations used: AHEI-2010, Alternative Healthy Eating Index–2010; aMED, Alternate Mediterranean Diet; CTS, California Teachers Study; DASH, Dietary Approaches to Stop Hypertension; PALEO, Paleolithic (index); SES, socioeconomic status.